Case Report

Postpartum pituitary necrosis: A report of 2 autopsy cases

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ABSTRACT

Major hormonal changes emerge during pregnancy. The pituitary gland is one of the most affected organs with altered anatomy and physiology. Sheehan's syndrome also known as Simmonds's syndrome or postpartum anterior pituitary necrosis is due to ischemic necrosis secondary to blood loss and hypovolemic shock after child birth. We report two autopsy cases of postpartum anterior pituitary necrosis with their clinical correlation. Histopathological examination of pituitary in both the cases showed extensive areas of coagulative necrosis involving the anterior pituitary. Both the cases were associated with indirect causes of maternal deaths like infectious etiology.

Key words: *Autopsy, Maternal Death, Pituitary, Sheehan's*

Sheehan's syndrome, first described by Sheehan in 1937, is a well-known cause of panhypopituitarism secondary to pituitary apoplexy [1]. This syndrome generally occurs after an intra- or post-partum bleeding episode characterized by severe hypotension or hemorrhagic shock. Vasospasm, thrombosis, and vascular compression of the hypophyseal arteries have also been described as possible causes of the syndrome. Although decreasing in frequency in recent years, it is still one of the most common causes of hypopituitarism in developing countries owing to the lack of effective management of postpartum bleeding [2]. During pregnancy, the pituitary gland undergoes remarkable changes in volume as a consequence of hyperplasia- a process triggered by placental estrogen secretion. However, the increased pituitary volume is not accompanied by a corresponding increase in vascular supply through the portal system since the pituitary gland cannot expand out of its bone cavity in the sella turcica. As a result, hypertension or vasospasm in the hypophyseal arteries, or an insufficient vascularization of the gland tissue, may irreversibly damage arterial circulation to the anterior pituitary. The posterior pituitary is usually not affected due to its direct arterial supply. Clinically, the patient presents with postpartum hemorrhage, shock, headache, and diplopia along with signs of anterior pituitary dysfunction in the form of failure to lactate and cessation of menses.

CASE REPORTS

We studied the histopathology of pituitary gland in 166 autopsies performed on deaths related to pregnancy over a period of 7 years from April 2011 to July 2017 and correlated with clinical findings. In all the autopsies in deaths related to pregnancy, pituitary was preserved in 10% formalin and hematoxylin and eosin stained sections were studied. We studied pituitary in 166 cases and found anterior pituitary necrosis in two cases (1.2 %).

Case 1

A 22-year-old, multigravida with 6 months amenorrhea came with chief complaints of bleeding per vaginum, fever with chills along with headache, breathlessness, blurred vision, diplopia, weakness, and vomiting since 10 days. She had a bad obstetric history in the past with delivery of a stillborn child. There was no history of gestational diabetes or eclampsia/preeclampsia. On general examination, the patient was febrile, conscious oriented, and her general condition was fair. Central nervous system examination revealed neck rigidity, the left lateral rectus palsy while other neurological signs were absent. Ultrasonography was suggestive of abruptio placenta. Computerized tomography scan brain shows meningeal enhancement with meningeal vessel congestion, moderate dilation of bilateral ventricles. The laboratory tests showed hypochromic microcytic anemia (hemoglobin 8.8 g/dL), low red blood cell count (3.06 × 106/μL), and low hematocrit levels (24.9%), total leukocyte count of 21,000/μL with neutrophilic leukocytosis, low sodium levels (108 mEq/L), and low chloride levels (83 mEq/L). Cerebrospinal fluid examination was within normal limits. The remaining tests were in the normal range.

At autopsy, on opening the skull, the brain showed massive cerebral edema and the pituitary was soft edematous. On histopathological examination, there were extensive areas of coagulative necrosis in anterior pituitary (Fig. 1), while posterior pituitary was intact showing nonmyelinated axons, capillaries, and nerve fibers (Fig. 2). Other organs on histopathological examination revealed bilateral bronchopneumonia. The cause

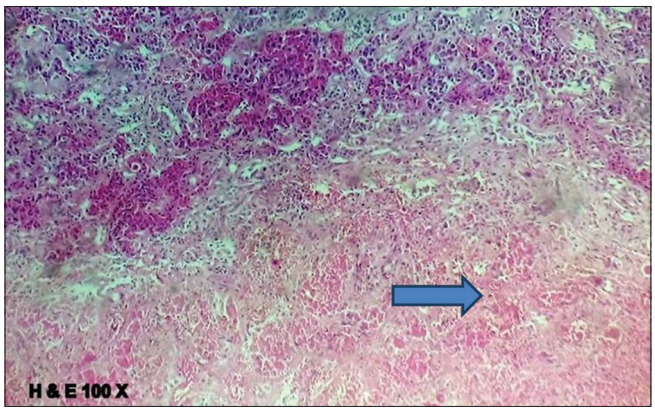

Figure 1: Extensive areas of coagulative necrosis in anterior pituitary (denoted by arrow) (H and E, ×100)

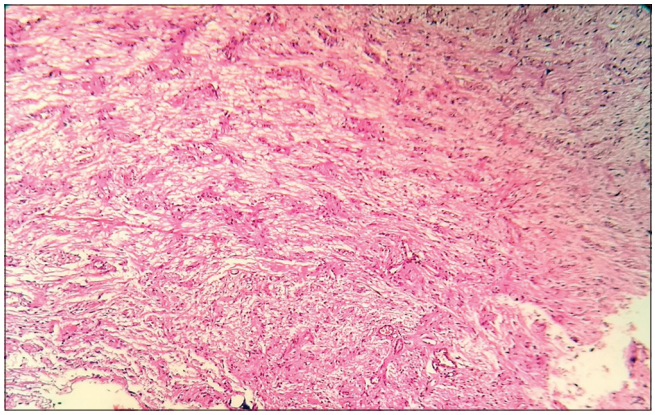

Figure 2: Posterior pituitary with nonmyelinated axons, capillaries, and nerve fibers (H and E, ×100)

of death, in this case, was ascertained as septic shock following bronchopneumonia.

Case 2

A 28-year-old woman multigravida presented with weakness, fever, cough, dyspnea, and postpartum bleeding per vaginum associated with abdominal pain and cramps in the lower limbs, preceded by persistent headache on the 8th day postpartum. In addition, the patient also reported lack of milk secretion after childbirth. Physical examination showed the altered level of consciousness and hypotension. The laboratory investigations revealed hypochromic microcytic anemia (hemoglobin 7.9 g/dL), and she had low sodium 106 mEq/L while other blood investigations were within normal limits. She had excessive bleeding in the course of her first delivery at the age of 23 years. At that occasion, she needed to be admitted to the intensive care unit and received blood transfusions. There was no similar case in her family history. The diagnosis of Sheehan's syndrome was established and pituitary magnetic resonance images demonstrated an empty sella. She received replacement therapy with L-thyroxin 100 µg/day and hydrocortisone 20 mg/day.

At autopsy, on opening the skull, the brain showed massive cerebral edema and the pituitary was soft edematous. On histopathological examination, there were extensive areas of coagulative necrosis in anterior pituitary, while posterior pituitary was intact showing nonmyelinated axons, capillaries, and nerve fibers. Other organs on histopathological examination revealed bilateral bronchopneumonia. At autopsy, the cause of death was ascertained as respiratory failure following bronchopneumonia.

DISCUSSION

Pituitary gland necrosis/Sheehan's syndrome is one of the most common causes of hypopituitarism in underdeveloped or developing countries in the postpartum period. Jashnani et al. [3] studied pituitary gland in in 37 cases and normal histology was noted in 34 cases; two cases showed infarction, out of which one was a case of intracranial hemorrhage in a hypertensive female and the other was a case of puerperal sepsis in a human immunodeficiency viral infection (HIV)-positive mother. Pituitary gland infarction is also known in HIV-positive individuals, which was noted in the only HIV-positive case in the above study. Collazos et al. [4] observed idiopathic pituitary necrosis in 10% HIV-infected patients at autopsy and thought to be due to direct effects of HIV, while we had bronchopneumonia and septicemia as the cause of death in both the cases. Hence, in our cases, there was hemorrhage and septicemia which could have contributed to anterior pituitary necrosis.

The patients described in our case report presented with symptoms of weakness, severe anemia and hyponatremia. The anemia that develops in Sheehan's syndrome is due to cortisol deficiency, hypothyroidism, and hypogonadism [5,6]. The other noteworthy clinical finding in our patients was hyponatremia. Hyponatremia is a common electrolytic disorder, occurring in 33-69% of all cases with Sheehan's syndrome [7]. The causative factors of hyponatremia in our patients could be due to volume depletion, cortisol deficiency, and hypothyroidism. In 1937, Sheehan reported 11 cases of women who died in the puerperium, all of whom had necrosis of the anterior pituitary gland-adenohypophysis. Nine of the 11 cases had severe hemorrhage at delivery [8]. Both the cases had bleeding in the form of abrutptio placenta or postpartum hemorrhage which eventually developed anterior pituitary necrosis. Infarction occurs secondary to the arrest of blood flow to the anterior lobe of the pituitary gland. Whether, this process results from vasospasm, thrombosis, or vascular compression is unclear. The fact that the posterior lobe of the pituitary is less commonly involved can be explained by the neurohypophysis's vascular supply arrangement which contains an anastomotic ring of blood vessels, which the adenohypophysis lacks [9]. In Sheehan's syndrome, inability to lactate after delivery due to prolactin deficiency and the development of amenorrhea from gonadotropin deficiency classically occurs. Our patient (Case 2), in agreement to documented literature, also could not lactate after delivery.

Hence, in our cases, multiple etiology apart from hypovolemic shock following antepartum and postpartum hemorrhage, both patients had septicemia along with bronchopneumonia which could have contributed to Sheehan's syndrome.

CONCLUSION

At autopsy, we found an incidence of pituitary apoplexy in 1.2% cases of all the maternal deaths. An index of suspicion should be kept in patients with hypovolemic shock due to antepartum or postpartum hemorrhage and patients who have symptoms such as headache and diplopia. Magnetic resonance imaging is a useful tool in diagnosing pituitary apoplexy. Both of the cases were associated with indirect causes of maternal deaths especially infections. Hence, adequate prenatal testing for these causes (infections), improving the nutritional status, sanitation can help in diagnosing this rare entity of pituitary and thereby lowering the maternal mortality rate.

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